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Fungous Disease Therapy

by Louis Schwartz, M.D.

Medical Director; Chief, Office of Dermatology, Industrial Hygiene, U. S. Public Health Service.

In last month's issue a review of the literature concerning fungous disease therapy was presented, with a detailed treatment of *tinea pedis* or ringworm of the feet. The discussion is continued on these pages with the treatment of *tinea capitis* or ringworm of the scalp.

Tinea Capitis

Duhring in 1883[2] recommended that *tinea tonsurans* be treated by manual epilation of diseased hairs and topical applications. Sulfur, ammoniated mercury, creosote, phenol mercuric nitrate, and iodine were used as fungicides. Glacial acetic acid or cantharides in collodion were painted once a week on the chronic patches and the aforementioned fungicides applied daily. He observed that this condition usually disappeared at puberty.

In 1899 Corlett[16] recommended that infected children be segregated or if this was not possible the scalp should be wrapped in impervious medicated dressings held in place by a skull cap. To be most effective the hair should be clipped close or shaved and the area manually epilated. Soap and water should be used to cleanse the scalp, which should then be dried and benzine applied and then one of the following parasiticides applied; phenol, 4-6 per cent.; copper oleate, 20 per cent.; mercuric oleate, 20 per cent. in vaseline; or formalin in 5-15 per cent. solution.

Roentgen ray and other treatments

Schamberg[3] reported in 1917 that Sabouraud had cured *tinea capitis* with one x-ray treatment. Manual epilation of the diseased hairs and the application of parasiticides was the method used by the former. As parasiticides, none of which was superior to any other, he employed betanaphthol 12 per cent., tar 35-50 per cent., chrysarobin 4-7 per cent., phenol 5 per cent., and oil of cade. Successful results were produced by perseverance and thoroughness in using the preparation.

Prophylaxis necessitating immediate strict isolation of the patient was stressed by Darier[17] in 1920. He cut the hair short every 8 to 10 days and painted the entire scalp every day with tincture of iodine. This was followed by an occlusion dressing of iodized petrolatum or chrysarobin ointment. Epilation by means of x-ray was his preferred method in those instances where x-rays were available.

Source of infection

Barber shops, combs, brushes and interchange of caps were blamed as the main sources of infection by Sutton[4] in 1927.

—Continued on page XXVII

Adapted from an address presented at the midyear meeting of the American Pharmaceutical Manufacturers' Association, Dec. 9, 1946.

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FUNGOUS DISEASE THERAPY

—Continued from page XXIII

Removal of the hair is necessary so that the drugs used can attack the parasites in the bottom and sides of the hair follicle. In addition to the preferred method with x-rays barium sulfide or frequent shaving of the head may be employed. Thallium acetate, although used in Mexico City was not considered as proper to use. The best fungicides according to Sutton are iodine, ammoniated mercury, chrysarobin, sulfur, tar, and betanaphthol.

Thus is summarized the advances made in the therapy of *tinea capitis* up to the beginning of World War II.

New developments

Livingood and Pillsbury[18] studied 105 cases of *tinea capitis* of the noninflammatory type as caused by *M. audouini.* In 27 per cent of the cases spontaneous cures without treatment occurred in 3 months to 2 years. They observed no consistently good effect from any one of the following fungicides; tincture of iodine, with and without 2 per cent. aerosols, iodine ointments with wetting-agent bases, phenyl mercuric nitrate, ammoniated mercury, sulfur and salicylic acid. They recommended x-ray epilation when available.

An epidemic of ringworm of the scalp began in the large eastern cities in 1942 and by 1943 smaller cities as far west as the Mississippi River reported epidemics. Investigations into the epidemiology and therapy of the disease were stimulated by this occurrence.

Estrogens

Daily oral doses of 5000 I.U. of estrone and daily application of an ointment containing 5000 I.U. per gram were used by Poth and Kaliski[19] on one series of 15 cases. A second series was treated with daily oral doses of 75,000 I.U. of diethylstilbestrol and daily application of an ointment containing 4000 I.U. per gram. Cures were affected in all but the 2 cases in which treatment was discontinued.

Effective results with sodium propionate were reported by Keeney and Brayler.[8]

Hagerstown, Maryland suffered an epidemic in 1944 and the U. S. Public Health Service was called upon to determine the method of spread and to formulate a method of control,[20, 20a]. This epidemic consisted of more than 600 cases or about 10 per cent. of the children in the city's grade school. It was controlled in one year by topical applications and was done without barring infected children from school or other public places. No x-ray epilation was used. Two years after the experiment began, all the children were cured except 15 who refused treatment.

Topical applications

It was found that the electric clippers in barber shops were the chief sources of infection. Seventeen different topical applications were tried, including zinc undecylenate and undecylenic acid, sodium propionate and propionic acid, copper oleate, colloidal iodine, a number of quaternary ammonium compounds, pentachlorphenol, phenyl mercuric salicylate, copper oleate, copper undecylenate, and salicylanilide. Copper undecylenate, and salicylanilide in a base of carbowax 1500 were the most efficacious remedies used. The children were treated daily in the schools by trained attendants who clipped the heads of infected children every 10 days.

X-ray epilation was preferred by McKee and colleagues[21] although they did cure 54 per cent. of their cases with trimethyl cetyl ammonium pentachlorphenate. Carrick[22] reported that no ointments should be considered as substitutes for x-ray epilation even though he had cured 41 per cent. of his patients with copper oleate, 40 per cent. with zinc undecylenate and undecylenic acid, 59 per cent, with sodium propionate and propionic acid, and all in about 130 days.

Value

In their textbook Lewis and Hopper[7] advocate manual epilation and application of 10 per cent. ammoniated mercury for small patches, x-ray epilation, and 3 per cent. ammoniated mercury for larger areas. In a later article[23] they conclude that no treat-

—Concluded on page XL

EDITORIALS

The Closed Shop and Monopoly in Medicine

We said in our January issue that "failure properly to integrate the general practitioner in our scheme of things would seem to signify a bankrupt civilization." In so far as he cannot be affiliated in some way with our hospital centers of continued education and training he tends to go "underground." This situation is essentially unwholesome, the more so because the implications of such a problem are sedulously evaded, just as though it had no reality; meantime much hypocritical stress is laid upon the indispensability of the general practitioner.

This situation has come about, we suppose, because formerly it was possible to care for many very ill persons in the home, in accordance with the general social pattern of things; now it is possible for many very ill persons to be cared for in hospitals —by diplomates; the general practitioner has not followed his patient into the "closed shop;" he does not belong to the "union."

To what extent the size and number of our hospitals have been intentionally or unintentionally limited in "scarcity economy" fashion is anyone's guess; in any case, society is now demanding more and larger hospitals. How do the unions expect to see them manned? It would seem that the displomate monopolists are due for some surprises.

Ironically enough, the beneficiaries of the medical closed shop are not, as a class, particularly sympathetic toward their counterparts in the labor movement. Logically, they ought to be staunch supporters of their industrial brethren.

The general practitioner will have to save himself; it manifestly won't be done for him; he will have to recapture the status enjoyed by his profressional ancestors, for it is in him that the aristocratic tradition of medicine is best exemplified. Can the great figures who created medicine be imagined limiting themselves in plebeian fashion —like unto the factory hand who performs one function on the assembly line year after year? It is perhaps logical, after all, that the medical plebeians of today should be unionized in closed shops.

Breasts and Brassieres

Commercial interests, obsessed by sexual fetishism centering around the mammary glands, are overemphasizing their product. At the same time they are not responsible for the original emphasis on the breasts in our time, for that started with the tight Hollywood sweater, inspired, no doubt, by some erotic male. The sweater, however, was not good enough for the purpose, since the breasts themselves were not usually sensational, so now the designers, artists, exploiters of fashions and manufacturers have given us a synthetic something that proclaims beguilingly to all bemused lechers: "Here I come; don't look now."

"Beguilingly" is the word, for these contraptions are intended to deceive the naive male. Beneath their phony opulence and aggression lie the undeveloped organs whose biological destiny has been thwarted by our civilization, along with all the other deficiencies so evident in our many undersexed women — pallid lips and roseless cheeks (under the lipstick, rouge and kalsomine), frigidity, sterility, dysmenorrhea, endometriosis, pelvic pathology incident to the congestion that regularly accompanies inability to experience unfrustrated intercourse, dystocia, etc. Of course, the baby seeking nourishment at the arid breast might as well suckle the brassière. And so artificial feeding becomes logical enough in a civilization decadent at so many points.

Those charged with the writing of the trade's advertising blah are clever; poets of

the mammary school, we should call them.

In fairness, though, we have to admit that our numerous weird sisters have their counterparts increasingly evident among the decadent males of the world, whose degeneracies have been accurately recorded by Conklin of Princeton University.

The New Type of Hypochondriac In Our Midst

Time was in this country when not many people were morbidly preoccupied with disease. One who was so preoccuped was the somewhat unique eccentric who was known as a hypochondriac.

But for one hypochondriac of those far-off days we now have morbidly preoccupied people by the thousands.

It is a communicable condition, but not normal to most cultures; and it is a new mass phenomenon here.

It has nothing to do with public health education of the stable elements in the population. There is nothing abnormal about the aims and results of such education, which is seen by such elements in non-neurotic, old American fashion. Public health matters, as such, do not interest the new type of hypochondriac; he bypasses them in favor of intimate personal items and doesn't give a thought to his town's sewage disposal system.

Nowadays one hears on every side discussions about the speakers' and their relatives' ailments in garbled medical versions. The public as well as private dining table seems to be the favorite place for these disgusting details.

These clinically absorbed people are further conditioned for commercial exploitation by radio and special advertising factors whose aims are alien to our older and better cultural standards. But these factors were never primarily responsible for the invasion and presence in our midst of basically morbid and neurotic elements with all their contaminating traits.

The Barbiturate Headache

The barbiturate habituation and poisoning problem is moving in as one of our major headaches. Dr. Charles Solomon of the Kings County Medical Society considers barbiturate habituation as serious a social menace as narcotic addiction. Colonel Garland H. Williams, Chief of the Eastern Division, Federal Narcotics Bureau, has pointed out that 90 per cent of the narcotic addicts also use barbiturates.

Contributing to this situation, in and out of hospitals, is the shortage of nurses. By 3 A.M. the sick are too fast asleep to need the attention of the relatively few available nurses.

Both State and local laws will have to be tightened up. Control of the barbiturates, thinks Colonel Williams, should be similar to that provided by existing narcotic regulations, instead of the weak, almost unenforceable present rules, which are naively based on good faith.

✚

Historical Division Transferred To Army Medical Library

The Historical Division of the Surgeon General's Office has reached a point in its work on the History of the Medical Department in World War II when its functions have been administratively determined to be more properly a division of the Army Medical Library than of the Surgeon General's Office. In order that the entire responsibilities of the Commandant, Colonel Joseph H. NcNinch, may be contained within one organization, the Historical Division has been transferred to and made a division of the Library.

This unit was set up in August 1941 under the direction of Colonel Albert G. Love to write a history of Medical Department activities in World War II. When completed the history will be issued in eight or more volumes covering the administrative, operational, clinical, and technical aspects of the Army Medical Department's wartime service in all theatres of operation. The Medical Department has a tradition in historical writing dating back to the time of the Civil War and was one of the first departments of the Army to undertake this type of publication.

A Rationale for the Treatment of Diabetes

George E. Anderson, M.D., F.A.C.P.

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With recent advances in knowledge regarding the fine points of carbohydrate metabolism,[1-2-3-4-5-6-7] it becomes necessary to "take stock" and apply the accumulated data toward a more rationalized therapy of diabetes mellitus. One question at this time must especially be pondered: "Is hyperglycemia with its attendant glycosuria *always* detrimental to the organism?" It is clinically obvious that hyperglycemia may, under certain circumstances in the *non-diabetic,* be so favorable to the well-being of the patient that it may with justification be considered physiological.[8-9-10-11-12-13-14-15-16-17-18-19-20] Among these states are parenchymal liver disease, the uremic state, degeneration of cardiac muscle associated with the anginal syndrome, the preparation of patients facing surgical crises, the protective influence of maintained relatively high glycemia against rupture of fragile retinal vessels in advanced arterioscleotic retinitis.[21] The opposite extreme, hypoglycemia,[22] certainly invites disaster in these respective diseases. It is, therefore, reasonable to suppose that diabetics under these same conditions would react favorably to moderate but measured hyperglycemia.

When one stretches this principle of liberalization, however, to apply to any and all diabetics, one is thereby discarding well-proven methods of therapy for a new and unproven approach, which has, as its main attraction, ease of application both for the patient and for his medical attendant.[24-25-26] The conservative physician has long had a "clinical hunch" that free diet and unlimited glycosuria are detrimental to the diabetic patient. When asked to prove his point, he has been at a loss because to date no one has ever been able to demonstrate that hyperglycemia per se directly causes any of the complications of diabetes save the relatively minor pruritus vulvae, enuresis, and faulty nutrition caused by the loss of vital nutrients through diuresis. On the other hand, no proponent of the "glycosuric paradise" has to date been able to state, categorically, that persistent unbridled hyperglycemia and glycosuria do *not* have a direct influence on the genesis of the more severe diabetic complications. The liberalists cite the point that retinitis and gangrene occur in the mild diabetic as well as in the more severe. They suggest that the same "anlage" which causes the diabetes, rather than the diabetes itself, causes the complications. One point, however, they cannot refute, that *without* the presence of a diabetic state, the commonly seen complications assume only the normal incidence for the age-grouping of the population. Anything, therefore, which favors the development or progression of the diabetes, likewise favors the incidence of the complications commonly seen in diabetes. If the cause of diabetes were completely established, its attendant morbidity through attack in this direction might be controlled. Failing this and pending more knowledge, the only safe approach at present is to control the diabetes itself in the hope of influencing its morbidity. This mode of attack is logical and clinically sound. Persistent hyperglycemia is unphysiological in that it is destructive of the "end-organ" in carbohydrate metabolism, the islets of Langerhans.[27] It therefore favors progression of the disease.

Experimental diabetes in animals has borne only superficial resemblance to spontaneous human diabetes. Actually, the removal of the entire pancreas in the human produces only a very mild diabetic state.[28] Pituitary-produced diabetes in animals, probably more than all others, resembles clinical human diabetes. Herein is sounded

Read before the 49th Annual Meeting of the Associated Physicians of Long Island at The Brooklyn Hospital, January 25, 1947.

the death knell to the unholy philosophy of unbridled glycosuria. Dohan and Lukens have recently found that steady hyperglycemia established in the normal cat and sustained over a 39-day period by peritoneal injection of 24 per cent glucose will produce a complete irreversible diabetes precisely identical with pituitary-produced diabetes.[27] They had previously found that in animals in which they had prevented hyperglycemia from developing by the use of restricted diet, phlorizin, or insulin, it was impossible to produce diabetes or typical lesions of diabetes in the pancreas by pituitary substance.[28-29] In other words, no hyperglycemia, no diabetes! In its early stages in the cat, pituitary-produced diabetes is reversible by anything which will correct hyperglycemia, namely, insulin, phlorizin, starvation, low carbohydrate intake. But, the human is not a cat; nevertheless, the human diabetic shows many of the characteristics of the Lukens cat. Early in the disease, just as in the cat, the diabetes is in part or totally reversible and by the same modalities as in the Lukens cat. This phenomenon of reversibility has been so striking in the mild obese adult type of diabetic that some authorities assert that the disease in these instances is not diabetes at all.[30-31] A blundering management over a lengthy period of time will frequently not preclude a considerable degree of *late* reversibility in these cases. That this is true is frequently demonstrated by the victim of the free diet who has had his disease sufficiently long to acquire one or more of the complications but who on rational conservative therapy will nevertheless promptly come under complete blood and urinary control (alas, often "after the horse has run away").

Feb. 1941 - F.M. - Male - 7½ yrs of age - Wt. 56½ lbs - Ht 50" - 26 K

Exton - Rose Test - 40 G. of glucose-repeated in ½ hour

Time	Blood Sugar	Urine Sugar
Fasting	89 mgm.	0
½ hr. after glucose	160 mgm.	0
½ hr. " 2nd "	211 mgm.	1.4%

(Theoretical Minimal Needs: 60 Cal per Kilo or 1560 Cal
Diet given Ch 160 -P 76 - F 70 - Cal 1569; P.Z.I. U3)

Exton - Rose Test - 5 weeks later (40 G. Glucose repeated in ½ hr.

Fasting	85mgm	0
½ hr after glucose	169 mgm	0
½ hr " 2nd "	190	0.8%

Mar 1945 Weight 80 lbs - Ht 57½ - Diet Ch 250 - P85 - F66-Cal 1938

Exton - Rose Test (10-10-45) (50 G. glucose repeated in ½ Hr.)

Fasting	90 mgm	0
½ hr after 1st glucose	164 mgm	0
½ hr " 2nd "	116 mgm	0

Fig. I. Pre-clinical Juvenile Diabetic. (Both parents diabetic)

The juvenile type of case presents a very different proposition. While reversibility—at least partial—is seen in this type of diabetic, it occurs only when treatment is applied vigorously along the most conservative classical lines and sufficiently early.[28] The liberalists completely "miss the boat" in this type of the disease, for "time is of the essence" and a relatively brief period of free diet and unlimited glycosuria will completely ruin the individual's chances for any degree of reversibility (the Lukens cat—subjected not only to pituitary influence but to an unremitting hyperglycemia as well, both diabetogenic; the cat will not develop the disease without a liberal diet).

Unfortunately, by the time treatment is initiated in most patients of the juvenile type, pancreatic damage is well advanced. These patients are usually not so fortunate as one young boy, aged 7½ years, who (Fig. I) first came under observation in 1941. Both of his parents were diabetic and accordingly they had been advised that all of their children would very probably at some time in their lives manifest evidence of diabetes. The parents routinely had been testing the child's urine and had on several occasions found heavy glycosuria without symptoms except fatigue. With the child having been on a full diet, an Exton-Rose glucose tolerance test showed: Fasting sugar 89 mgms.; 30 minutes after 40 G. of glucose, 160 mgms.; 30 minutes after a second 40 G. of glucose, 211 mgms. with a glycosuria of 1.4 per cent, a mild but prolonged curve without Staub-Traugott effect. The test was repeated for confirmation. The child weighed 58 lbs. (26 K.). Theoretical minimal caloric needs at 60 calories per kilo were 1560 calories. Diet given: Ch 160—P 76—F 70—Cal. 1569. Protamine zinc insulin U 3 was given daily. Insulin in minute dosage was kept up until January 1944 when it was discontinued. The diet had gradually been stepped up to: Ch 250—P 85—F 66.

Growth, weight-gain, and sense of well-being had been satisfactory. Two years after stopping insulin the Exton-Rose test (using 50 G. glucose, repeated in 30 minutes) showed a perfectly normal curve with normal insulin-sensitivity and absence of glycosuria. It is now five years since this child first came under observation. He is rugged, healthy, and of course, not on insulin. He is completely aglycosuric on a diet only qualitatively restricted. *Comment:* What would have been this child's lot, had he been encouraged to follow his own inclinations regarding sugar, candy, and pastry? Obviously, just one more juvenile diabetic!

As in the Lukens cat, clinical diabetes starts in the guise of an excessive demand on the part of the organism for increased insulin production.[27-28-29] Be this demand in the nature of excessive caloric ingestion, excessive carbohydrate intake, or a hyperfunctioning pituitary gland, the inordinate insulin demands put the insulin mechanism to extremes of overproduction.[32] Subsequently there is added to this excessive and fatiguing demand for insulin a decreasing ability on the part of the mechanism to produce even enough insulin to meet ordinary normal demands much less the exaggerated requirements;[33-34] or if the pancreas is still able to produce in adequate quantity, a state of resistance or insensitivity on the part of the organism to insulin supervenes, producing the end result of a relative insulin insufficiency. This was mathematically demonstrated by Himsworth, who pointed out that insulin effect is less a matter of quantity of insulin than it is a function of the degree of sensitivity of the organism to insulin.[35] As a result of insulin-resistance, both endogenous and exogenous insulin suffer from the law of diminishing returns, for a given amount of insulin no longer has the anticipated effect. That insulin-sensitivity plays an important role in insulin efficiency is further indicated by the fact that with insulin unburdening and subsequent adequate stimulation of the insulin-mechanism by diet, a return to normal insulin-sensitivity can frequently be effected.[36]

How may one apply these facts in treating the average diabetic patient? While all treatment must be individualized, the first step in treatment should be an attempt to reduce the excessive insulin demands.[37] The second step should be an effort to help the individual meet the minimized demands as adequately as is possible. Later, after the establishment of complete chemical control, it will frequently be possible to stimulate increased "returns" on insulin (both exogenous and endogenous) or grams of carbohydrate utilized per unit of insulin, by the "stimulating" effect of a *relatively* liberal carbohydrate intake.

How shall one accomplish the first step, that of reducing inordinate insulin demands? There is at present only one sure means of doing this, namely, by cutting the total caloric intake of the individual down to a point *just at or slightly but painlessly below* his actual caloric needs. It is to be emphasized that such restriction is a temporary measure, a means of unburdening the intrinsic insulinogenic mechanism of its overwhelming load, persistent hyperglycemia, and thus preparing it for active function on its own. A diet calculated at 25 calories per kilo at the normal actuarial weight for the individual will usually accomplish this end. This regimen is a far cry from the old starvation routine of the pre-insulin era.[33-39] However, the truths established by the great trail-blazer in this field, Doctor F. M. Allen, are still valid even when applied in painless moderation. An overloading dietary with insulin to meet the excessive needs will not result in improving the function of the patient's own intrinsic insulin mechanism. There must be at least mild caloric restriction. The patient may neither store fuel nor lose it in excess if he would recapture his native tolerance for carbohydrate. It is also obvious that the carbohydrate intake at this leveling time should be an amount not calculated to increase the insulin demands. A fairly neutral level in this respect is between 100 and 150 grams daily (Fig. II).

Frequently under such mild caloric restriction with carbohydrate limitation, the patient will become completely aglycosuric; if not, insulin must be administered to accomplish complete chemical control, for

this is the first major hurdle to be jumped in treatment.

Having accomplished this end, the principles (Fig. III) enunciated by Himsworth regarding the "stimulation" of normal insulin-sensitivity are brought into the picture.[35] In *normal* individuals, diets containing less than 100 Grams of (Fig. II) carbohydrate in 24 hours tend to be "non-stimulating" of insulin sensitivity; those under 50 G., to be actually depressant to the mechanism; those between 150 G. and 200 G. are markedly "stimulating;" those between 200 G. and 350 G. in 24 hours are somewhat less "stimulating;" between 350 G. and 450 G. there

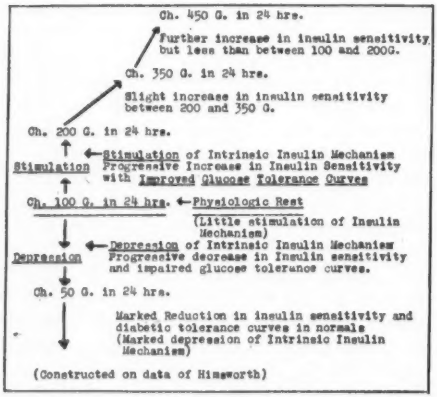


Fig. II. Himsworth (Ref. 35) has shown that in* normals *increased or decreased sensitivity to insulin in response to dietary carbohydrate corresponds exactly with measured quantitative improvement in the individual glucose tolerance, suggesting that sensitivity to insulin rather than quantity of insulin available is in the* normal *the more determinant factor in carbohydrate tolerance.

is another sharp wave of "stimulation" but definitely less than between 150 G. and 200 G.

In The Brooklyn Hospital Diabetes Clinic, Anderson *et al.*[36] have repeatedly demonstrated that this same "stimulating" effect to normal insulin-sensitivity, which increases insulin efficiency unit for unit, occurs *in the diabetic only* when the total calorigenic intake is sufficiently low to permit of adequate chemical control without insulin extravagance—usually at a point just *at* or *slightly* below the individual's actual caloric needs.* In other words, as a corollary, in the calorically overloaded diabetic individual, the normal "stimulating" effect of a relatively high carbohydrate ingestion is supplanted by an overwhelming depressant effect as this is translated into reduced carbohydrate tolerance, reduced insulin sensitivity (increased insulin resistance) and consequent progressive extrinsic insulin needs.

Accordingly, the clinician has within his grasp the ability to establish relative rest of an overtaxed insulin mechanism by reducing the total calories ingested and holding carbohydrate within the relatively "non-stimulating" range. As soon, however, as chemical control has been accomplished, the "non-stimulating" routine may safely give way to a "stimulating" regimen, raising the carbohydrate of the diet at the expense of the fat but maintaining the same caloric (equicaloric) value of the diet. With chemical control of urine and blood having been established, and with a diet in the range of 200 ± G. of carbohydrate, insulin-sensitivity now definitely increases. *Pari passu* with this improved sensitivity to insulin, there develops increased efficiency in the patient's carbohydrate utilization in relation to insulin, grams of carbohydrate utilized per unit of insulin. The initial extrinsic insulin requirements as these may have existed are accordingly reduced. In fact, the efficiency of both extrinsic and intrinsic insulin will frequently so improve that the carbohydrate intake may now be raised up to 250 Grams or more without added insulin, provided this increase is made at the expense of the fat of the diet while keeping the caloric value constant. Such improved insulin efficiency is the herald of a more physiologic state.

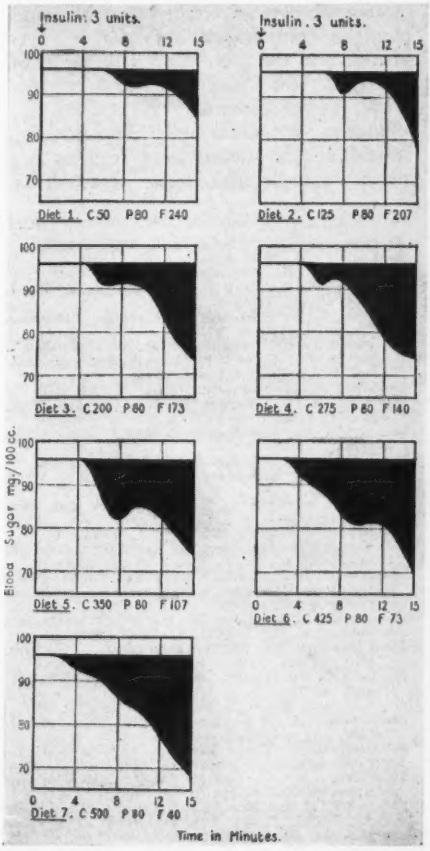
The next procedure is to step up the caloric value of the diet by means of fat additions to a point adequate to meet the patient's daily needs as these may be determined by the scales and sense of well-

*** Some will attribute such apparent improvement in insulin sensitivity to fatty infiltration of the liver secondary to the restricted food intake. This is refuted by persistence of the improved insulin efficiency even after the diet has been raised to the optimal needs of the patient.**

being of the individual. If done in step-ladder fashion, such increase in calories can frequently be accomplished without increased insulin administration provided the patient is at no time overloaded in calories.[36] Emphasis is placed on the "before meal" urine specimens being maintained sugar-free. It is understood that with the higher carbohydrate values of the diet, there may be brief periods of transient post-prandial spill-over analogous to that of an exaggerated alimentary glycosuria. Such spill is not projudicial to improved carbohydrate tolerance *provided* there are phases of complete recovery before the next meal and especially complete recovery to normal glycemia through the night, the post-absorptive period. Nocturnal glycosuria is not allowed under any circumstances. With adequate complete recovery phases, transient brief periods of hyperglycemia are compatible with improvement in carbohydrate tolerance and improved insulin efficiency—both of which are readily measurable. In fact, such controlled blood-sugar fluctuations are in themselves probably more conducive to improved function than is the deliberately attained flat 24-hour curve.[40] Recovery phases must, however, be insisted on, lest the clinical diabetic through persistent hyperglycemia become analogous to the Lukens cat with progressive islet degeneration.

In the over-all picture there are encountered in the essayist's experience in the teaching clinic of The Brooklyn Hospital approximately only four per cent of presumably *early* diabetics in whom, despite the most meticulous dietary care and insulin management, one cannot establish adequate chemical control. These four per cent of patients, for the most part insulin-supersensitive juvenile types of diabetes, are the so-called "brittle cases" in whom adequate insulin administration results in extreme fluctuations between dangerous hypoglycemia and marked hyperglycemia—a state of instability incompatible with normal existence either for the patient or for his medical attendant. One reluctantly but advisedly surrenders these patients to a more liberal type of management, sacrificing thereby almost all hope for any degree of functional recovery as this is measured by reduced insulin needs. However, in the other ninety-six per cent of cases, it is unconscionable not to give the patient an opportunity to approach a more normal physiology, even to do without exogenous insulin if at all possible. Since "trial and error" has proven to be the

Fig. III. From Himsworth, H. P., Clinical Science 2:67, 1935. The shaded areas indicate the individual's blood-sugar fall in response to intravenous crystalline insulin after having been on the diet noted under ach figure (all diets equicaloric). Note the marked increase in shading after the diet Ch.200 as compared with that after diet Ch.125 and the relatively insignificant increases after diets Ch.200 until much higher figures are reached.



only certain method of determining which patients will fall into the four per cent category, it is incumbent on the medical attendant to initiate conservative treatment in all cases and vary this in accordance with experience.

Irreversibility is analogous, as Mosenthal[37] has so aptly pointed out, to the finished product in alloxan diabetes.[37] It would seem unwarranted in the fully established state of irreversibility to insist on the same standards of chemical control demanded in the reparable stages of the disease. In accordance with Mosenthal's and Handelsman's thoughts in this respect, the dietary of these patients is more liberalized.[41] The warning of Ricketts, however, should be borne in mind that the innocuousness of hyperglycemia from the standpoint of complications has not yet been proven, and the conscientious doctor must, therefore, still "play ball" and not surrender to the extreme.[42]

Insulin is consumable and therefore should not be wasted by a fruitless persistent hyperglycemia. Since persistent hyperglycemia is destructive of the "end-organ" in carbohydrate metabolism, the pancreas, it should be avoided, whatever may or may not in the future be proven regarding its innocuousness in relation to the more serious complications of the disease. Whatever may ultimately be established in regard to whether the pancreas is of primary or of secondary importance in the genesis of diabetes, one fundamental truth stands, *without insulin normal carbohydrate metabolism in the human breaks down;* accordingly, whatever it may be, infection,[43] proteolytic enzymes,[44-45] overacting pituitary,[27-28-29-33-34-46-47-49] superalimentation, or *persistent* hyperglycemia,[27-28-29-38-49] anything which finally impairs the ability of the pancreas to make insulin or which extravagantly pushes the insulin mechanism beyond its productive capacity and into a state of chronic fatigue and "bankruptcy" is compromising normal physiology. The possibility of *any* degree of reversibility in the diabetic demands that the clinician provide for adequate periods of blood-sugar recovery.

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Pulmonary Tuberculosis

Modern Concept and Recent Advances in Treatment

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V*

The serous membranes of the body, the meninges, the pericardium, and the peritoneum, are particularly liable to tuberculous disease. The pleurae are not an exception to this rule. In fact, we believe tuberculosis of the pleurae is as common as tuberculosis of the lungs. In all forms of tuberculosis the morbid processes extend from the pulmonary parenchyma to the visceral pleura. Its anatomical relation, its blood supply and its lymphatics render the pleura peculiarly liable to infection with tubercle bacilli. The visceral pleura is very thin and firmly adherent to the lungs. It rests on a layer of areolar elastic tissue and is continuous with the elastic and connective tissue of the lung. The blood supply of the visceral pleura is derived from the bronchial and pulmonary arteries. The parietal pleura receives its blood supply from the internal mammary, intercostal, phrenic, mediastinal and bronchial arteries. So, we can readily see that inflammatory processes of the lungs, when extending to the surface, will implicate the visceral pleura, while the parietal sheet will only be affected by contact. The visceral pleura is very rich in lymphatics. These are especially numerous in the interlobar fissures. A connection with the bronchial glands can be easily demonstrated by the fact that with age they show darkening, due to carbon particles inhaled into the lung, just as the bronchial glands do. The lymphatics of the parietal pleura pass to the small intercostal glands situated beneath the heads of the ribs and through this connection with the lymphatics of the fourth or fifth intercostal glands they are connected with the axillary glands. There are also communications between the lymphatic systems of the chest and the abdomen through the anastomosis between the lymph vessels of the pleura and those of the peritoneum, particularly in the case of the lymphatics covering the lower surface of the diaphragm. Thus we can see that the pleural space, though nominally a closed sac, is closely connected with the lungs and the abdomen. The pleurisies are divided into three main groups:

1. Initial or idiopathic pleurisies.
2. Pleurisies occurring in acute pulmonary tuberculosis.
3. Pleurisies that occur in the course of chronic pulmonary tuberculosis.

These pleurisies may be moist or dry, serous or serofibrinous, serosanguineous, or frankly purulent.

To consider all idiopathic pleurisies as of tuberculous etiology is to lay oneself open to argument. However, if we exclude pleurisies that occur a) in the course of rheumatic infections, b) those that follow postpneumonic conditions, especially when sulfonamides are used, c) and those that are caused by trauma, we find that 90 per cent of the effusions that we have had a chance to observe for periods varying from six months to four years eventually prove to be tuberculous.

The following case, briefly reviewed, is not exceptional. The time interval between the onset of the pleurisy and the final manifestation of pulmonary tuberculosis was a little over three years.

Case I. Mrs. Natalie K. came under our care on December 26, 1942. There was a history of illness ushered in by chills, fever, malaise, and pain in the left side of the chest. She was treated by the family physician for three weeks. During this period, she was given large doses of sulfonamides. The pleuritic pains subsided but the elevation of temperature continued. When

* Part V of a series.

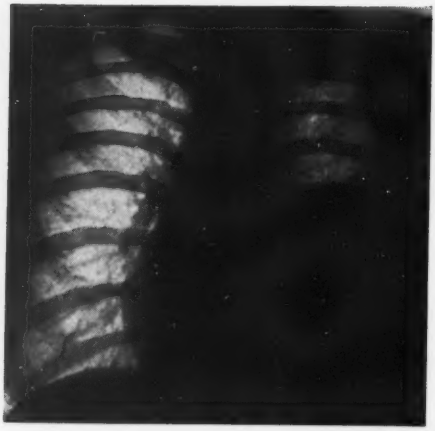


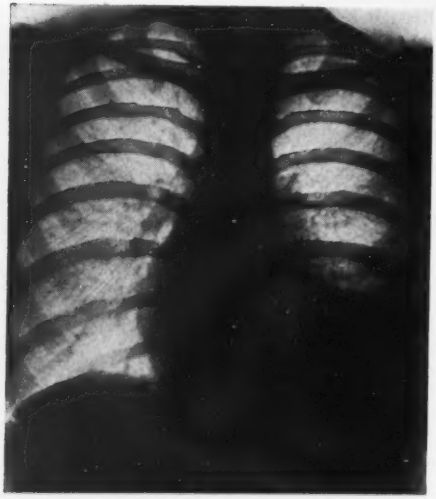
Fig. 1. Case I. Natalie K., 12/26/42

Shows a pleural effusion covering the lower two-thirds of the left pulmonary field.

admitted to the hospital, physical examination confirmed by x-ray showed a pleural effusion occupying the lower two-thirds of the left pulmonary field. (Fig. 1). We aspirated a little over 500 cc. of clear, straw-colored fluid. Cytological examination showed only an occasional small lymphocyte. Direct smear showed no tubercle bacilli. Culture of the fluid showed no growth of any pyogenic organisms. A guinea pig was inoculated with the aspirated material, but no tuberculosis was found in the animal at the end of six weeks' time. An x-ray taken soon after the second aspiration of nearly 500 cc. showed no evidence of tuberculosis in the left pulmonary field. (Fig. 2). Yet on February 4, 1946, this woman showed signs of an acute tuberculous exudative lesion in the left upper lobe. (Fig. 3).

Fig. 2. Case I. Natalie K., 12/29/42

Shows after the removal of 500 cc. of clear, straw-colored fluid an apparently normal lung. Except for a slight haziness on the left base (due to a small amount of residual fluid), there is no evidence of parenchymal tuberculous pathology.



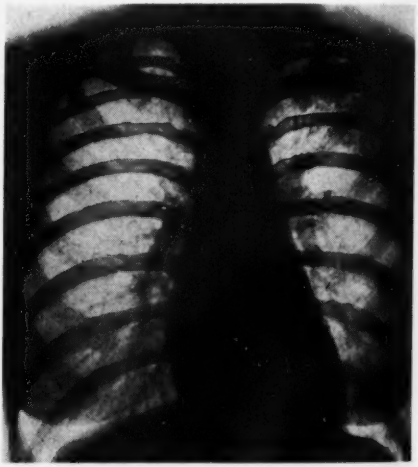


Fig. 3. Case I. Natalie K., 2/4/46

Shows definite evidence of the development of an acute exudative tuberculous lesion in the left upper lobe. This became apparent nearly three years after the initial pleural effusion.

In many instances of pulmonary tuberculosis that we see which appear suitable for treatment by artificial pneumothorax, failure to find a free pleural space often elicits a similar story from the patient. They vaguely recall that some time in the past they had an episode characterized by fever, chills, and pain in the chest. Then there was a subsidence of symptoms, especially the pleuritic pains. Then a longer or shorter period of apparent good health.

Finally the development of signs and symptoms of pulmonary tuberculosis.

It is our impression that these transient pleural effusions are manifestations of a primary complex that is localized in the pleura and is analogous to the primary complex that we often see in the lungs.

Another form of pleurisy with effusion that is of great interest to us is that occurring in the course of the treatment of pulmonary tuberculosis by means of artificial pneumothorax. Though the patient has been getting along well, there will occur at times following a refill a sudden rise in temperature and sharp lancinating pains on the side of the chest that is being treated. Examination will reveal the presence of fluid in the pleural space. Though these abrupt onsets are seen frequently, as a rule these effusions will form gradually without producing any symptoms.

Statistics as to the frequency of occurrence of effusions in the course of pulmonary tuberculosis treated by pneumothorax vary a great deal. However, we find that all cases treated by pneumothorax show the presence of fluid in the pleural space at some time or other. It may be only a minute amount noted in the costophrenic sulcus or it may be sufficient to cover the dome of the diaphragm.

The control of these effusions has been the cause of a great deal of research. Calcium chloride given intravenously has been advocated for the prevention of such effusions. We have not had any experience with this agent. We find as a rule that these effusions are transitory in character and will disappear in a few weeks or at the most a few months. Frequently, however, in spite of repeated aspirations of the pleural exudate and replacement of the fluid with air, the fluid will continue to increase. The lymphocytes found in the effusions will be replaced by polymorphonuclear leukocytes. The cellular elements will increase rapidly. The specific gravity and the albumin content of these effusions will become disproportionately high.

The danger of these large effusions is well known to those who treat pulmonary tuberculosis. The increase in intrapleural pressure may cause herniation of the mediastinum, and, in some cases, rupture of the "weak spot" in the mediastinum has been reported. Displacement of the heart and cardiac embarrassment often result. At times re-expansion of the lung beneath the fluid level will occur, thus terminating a previously very effective collapse.

Most observers have come to the conclusion that when effusions increase beyond a certain limit and cause pressure symptoms or an elevation of temperature, frequent aspiration and the replacement of the fluid by air is essential. But when these effusions become purulent, active measures must be instituted to cope with the situation.

Pleural effusions occurring in the course of the treatment of pulmonary tuberculosis by artificial pneumothorax can be classified as:

1. Simple, or benign; only noted in the course of fluoroscopic or x-ray examinations.
2. Tuberculous pleurisies.
   a. Serofibrinous, readily absorable.
   b. Chronic serofibrinous, absorbed slowly or not at all.
   c. Purulent, due to secondary invasion of saprophytic organisms.
3. Septic pleurisies resulting from general infection.
4. Those due to pulmonary perforations.

For the conservative treatment of these pleural effusions, various agents have been used and advocated—irrigation with salt solution—with various dyes such as methylene blue and gentian violet. In our hands, these various agents have not proven successful.

In the prepenicillin era, we often resorted to the instillation of metaphen in oil or aqueous solution of metaphen into the pleural space to control the effusions that showed a tendency to become purulent.

The following cases are cited to show the mode of onset, the underlying pathologic process, and the interminable length of time that it took to control and eradicate these effusions.

Case II. A case of pleural effusion occurring in the course of acute pulmonary tuberculosis that rapidly turned into an empyema.

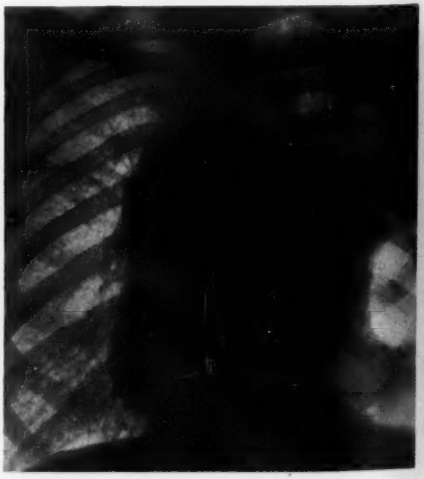
Fig. 4. Case II. Mr. S., 4/27/32

Pleural effusion completely blocking out the left pulmonary field with marked displacement of the heart to the right. Aspirated material showed thin, turbid, greenish-yellow exudate.

Mr. S., thirty-four years of age, was taken ill April 1, 1932. He complained of sharp, lancinating pains in the left side of the chest, slight cough, very little expectoration, temperature 102° to 103°, and marked dyspnea. On April 26, 1932, he was admitted to the hospital. Physical examination revealed dullness throughout the left side of the chest. Breath sounds feeble and distant in the upper third of the chest. Fine crepitant râles in this area. No breath sounds audible in the rest of the left pulmonary area. Heart evidently displaced to the right. Patient appeared to be extremely ill. Physical examination was done under difficulties as the patient was too ill to cooperate. X-ray examination on April 27, 1932, showed complete blocking out of the left pulmonary field with marked displacement of the heart to the right. (Fig. 4). That same day one thousand cc. of thin, turbid, greenish-yellow exudate was removed. The patient's breathing became less labored, cyanosis less marked; temperature remained unchanged. Examination of the sputum showed tubercle bacilli Gaffky X. He continued to be toxic, sweating profusely. A second aspiration and the removal of another thousand cubic centimeters of pleural exudate was done on May 2, 1932. The aspirated material now showed a complete change in appearance. It was now definitely purulent. X-ray examination of the chest at this time showed an acute exudative lesion in the left upper and lower lobe with several shallow, thin-walled cavities. (Fig. 5). Following this aspiration, two hundred cc. of air was given by means of the pneumothorax apparatus. Initial reading was plus ten, plus eight. Final reading, plus twenty-two, plus eighteen, and 25 cc. of metaphen in oil was instilled in the pleural space. Within a week's time the temperature subsided and remained normal until the patient was discharged from the hospital. The pneumothorax space was limited in extent. It was really an air pocket from the fourth to the seventh rib in the midaxillary line. Aspiration, replacement with air, and the instillation of varying amounts of metaphen in oil, 1:5000, at the end of each refill were continued at two-weeks'

Fig. 5. Case II. Mr. S., 5/21/32

After second aspiration, x-ray revealed an acute exudative tuberculous lesion in the left upper and lower lobes with several shallow, thin-walled cavities. Aspirated material was now definitely purulent in character.



intervals until January 29, 1933, when the fluid became thin and was found to be barely covering the dome of the diaphragm. (Fig. 6). Physical examination now revealed a few moist râles in the left upper lobe at the end of expiratory cough; sputum negative for tubercle bacilli; no clinical or roentgenographic evidence of cavitation. Patient gained forty pounds in weight. Pneumothorax, aspiration of the exudate and the instillation of metaphen in oil were continued until November 5, 1933. The lung gradually re-expanded and final absorption of the remaining fluid took place.

He returned for examination on January 18, 1937. The x-ray report is as follows (Fig. 7):

"Comparison with the examination made February 9, 1933, shows complete re-expansion of the left lung. The infiltrative lesion has been replaced by fibrotic tissue. The left diaphragm is somewhat obscured by pleuritic adhesions. The heart is retracted to the left, probably because of cicatricial contraction. There is no evidence of activity."

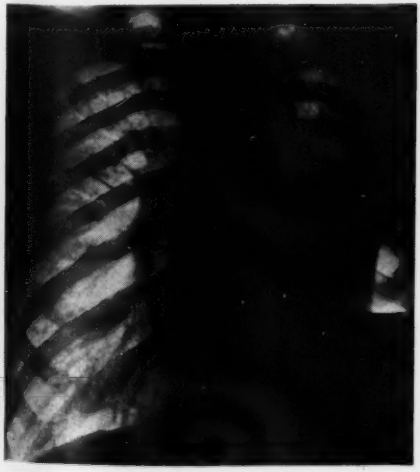


Fig. 6, Case II. Mr. S., 2/9/33
Shows the presence of a small pneumothorax on the left side and a fluid level just barely covering the dome of the diaphragm. Fluid at this time thin and is aspirated with little difficulty. Pneumothorax refills were discontinued at this time, but aspiration of the exudate and the instillation of metaphen in oil 1:5000 continued until 11/5/33, when the lung gradually re-expanded and final absorption of the remaining fluid took place.

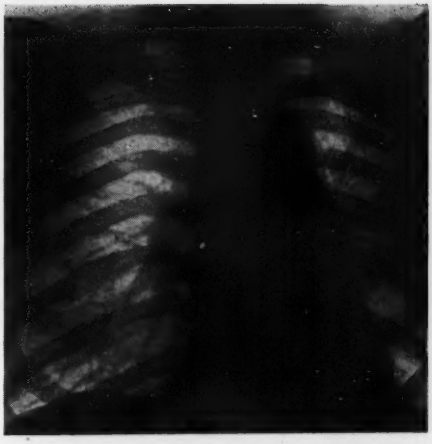


Fig. 7. Case II. Mr. S., 1/18/37
Comparison with examination made 2/9/33 showing complete re-expansion of the left lung and an apparent obliteration of all the cavities. The infiltrative lesion has been replaced by fibrotic tissue. The left diaphragm is somewhat obscured by pleuritic adhesions. The heart is retracted to the left, probably because of cicatricial contraction. There is no clinical or roentgenographic evidence of activity at present. At the end of 1933 the patient resumed his former occupation as undertaker, and is retaining the weight he gained when all treatment was stopped 11/5/33.

Case III. An asymptomatic type of empyema. Exemplifies the gradual change from a simple effusion into an empyema occurring in the course of pneumothorax treatment.

History of Mr. G. begins in July, 1930. At that time physical examination confirmed by x-ray showed an acute exudative lesion in the left apex and subclavicular region. Pneumothorax treatment was suggested but refused and expectant treatment was continued until September 12, 1931. This time examination revealed a fairly large cavity in the left upper lobe, with marked activity throughout the left pulmonary area. (Fig. 8). Artificial pneumothorax was instituted October 9, 1931, and about 40 per cent collapse of the left pulmonary field was obtained. (Fig. 9.) The x-ray revealed multilocular cavitation in

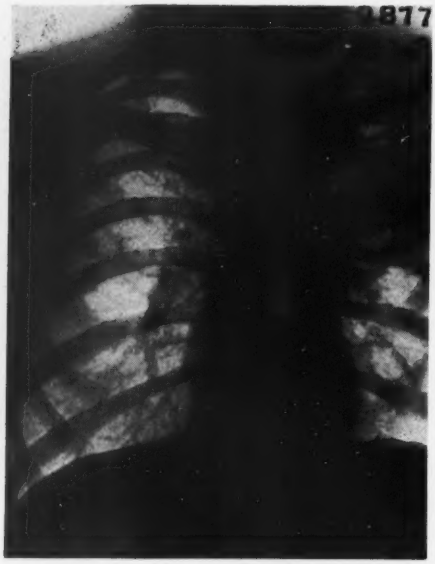
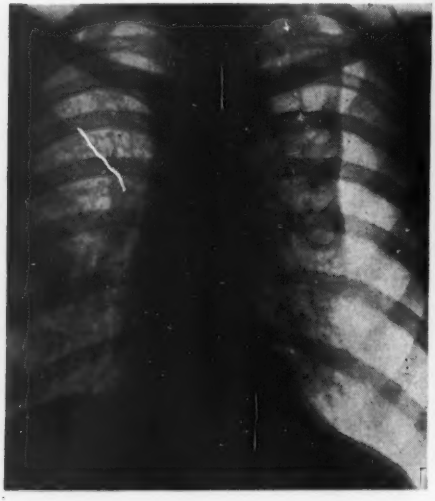


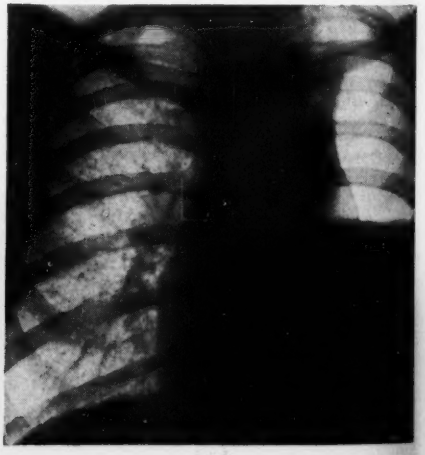
Fig. 8. Case III. Mr. G., 9/12/31
Shows extensive fibrocaseous pneumonic process involving the left upper lobe with large vomica in the left apex.

Fig. 9. Case III. Mr. G., 10/29/31
Shows artificial pneumothorax on the left side with about 40 per cent compression of the left pulmonary field, and multilocular cavitation in the left upper lobe.



the partially compressed lung. On May 7, 1932, a pleural effusion was noted. (Fig. 10). Although this was asymptomatic, that is, no local or constitutional effects were noted, in the weeks following the development of the effusion the patient began to lose weight and "tired feeling" became marked. The effusion reached the level of the fourth rib anteriorly and aspiration was decided upon. The aspirated material revealed a frankly purulent effusion. Aspiration, continuation of the pneumothorax treatment, and the instillation of metaphen

Fig. 10. Case III. Mr. G., 5/7/32
Shows a pleural effusion on the left side reaching to the level of the fourth rib in the midaxillary line. Apical cavity only partially compressed. Cavitation in the rest of the left pulmonary area apparently obliterated. Although this effusion was asymptomatic, in the weeks following the development of the effusion, the patient began to lose weight and "tired feeling" became marked. Aspiration, continuation of pnumothorax treatment, and the instillation of metaphen in oil 1:5000 at the end of each refill were begun.



in oil 1:5,000 at the end of each refill were begun. In a short while there was a return to gain in weight and a feeling of well-being. In May, 1933 (Fig. 11), the patient began to run a hectic course with increase in cough and expectoration. The sputum again became positive for tubercle bacilli. X-ray revealed a fibrocaseous process in the right upper lobe with a cavity in

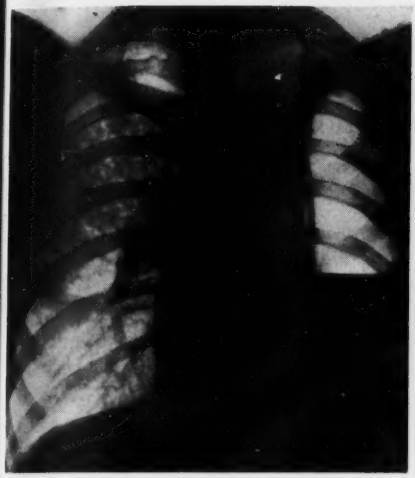
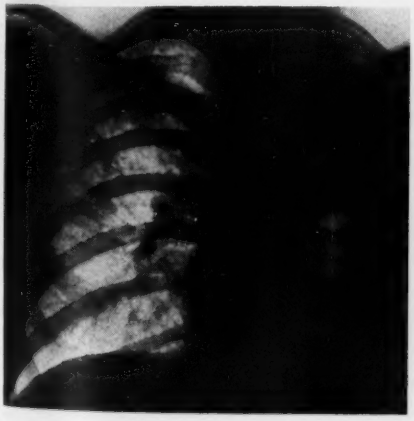


Fig. 11. Case III. Mr. G., 5/16/33

In May, 1933, the patient began to run a hectic course with increase in cough and expectoration. The sputum again became positive for tubercle bacilli. X-ray revealed a fibrocaseous process involving the right upper lobe with a cavity in the second right interspace near the periphery of the lung.

Fig. 12. Case III. Mr. G., 9/12/33

The left lung was allowed to re-expand somewhat. The fluid in the left pleural space increased rapidly, practically filling the left side from apex to base. A phrenicectomy was performed on the right side.



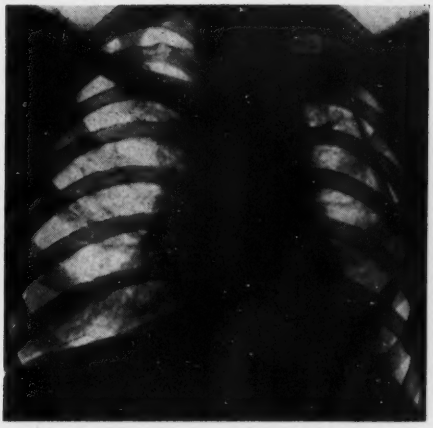
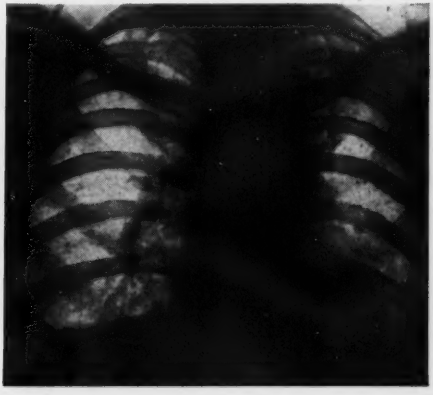


Fig. 13. Case III. Mr. G., 10/6/36

The final clearing up of the empyema occurred in September, 1935. This x-ray film, taken 10/6/36, shows a ten to fifteen per cent compression of the left pulmonary field and obliteration of all cavitation. This plate also shows a small pneumothorax on the right with disappearance of the cavity in the second right interspace and marked improvement in the general pathology on the right.

Fig. 14. Case III. Mr. G., 12/28/44

Shows a residual thickening of the pleura in the left base. The heart and mediastinum are slightly shifted to the left. There are scattered calcific nodules in both pulmonary fields. The left apex shows slight pleural thickening. The right hemidiaphragm is slightly elevated. There is no clinical or roentgenographic evidence of active tuberculous disease.



the second interspace near the periphery. The left lung was allowed to re-expand somewhat, and pneumothorax treatment on the left side was temporarily discontinued. The fluid in the left pleural space rose quite rapidly, practically filling the left side from apex to base, (Fig. 12). A phrenicectomy was performed on the right side with marked improvement in the clinical picture and the disappearance of the cavity on the right. Treatment on the left side was reinstituted. The exudate gradually became thinner and finally almost complete absorption took place. On or about September 25, 1935, the left pleural space became dry. Although following the phrenicectomy on the right there was marked improvement in the patient's condition, activity persisted and the sputum continued to be positive. It was then decided to institute artificial pneumothorax on the right side and on October 6, 1936, the first treatment was given (Fig. 13). Thus the case became one of bilateral pneumothorax. Following this procedure, the sputum became negative and the patient gained in weight. Six months later this patient felt well enough to resume his former occupation—that of bookkeeper for a local manufacturing concern. Pneumothorax refills were stopped February 21, 1943. A check-up in this case done on December 28, 1944 (Fig. 14) shows a residual thickening of the pleura in the left base. The heart and mediastinum are slightly shifted to the left. There are scattered calcific nodules in both pulmonary fields. The left apex shows slight pleural thickening. The right hemidiaphragm is slightly elevated. There is no clinical or roentgenographic evidence of active tuberculous disease.

It is not our intention to oversimplify the rationale for the treatment of pleural effusions. It often requires the exercise of all the acumen and ingenuity of the physician to decide what procedure is best to follow. It is not a rule-of-thumb regimen that we outline. To decide when a pleural effusion is to be allowed to absorb without interference and when intervention is necessary, we have used the following criteria:

(1) Thoracentesis and the removal of 10 to 20 cc. of pleural fluid. We note the color, clarity, or turbidity of the aspirated material.
(2) Cytological examination — cell count and differential stain.
(3) Bacteriological studies.

We adhere to the dictum that all pleural effusions are to be considered tuberculous unless proven otherwise.

In those effusions where no definite evidence of tuberculous etiology can be established, we allow them to absorb.

When lymphocytes predominate, we consider such effusions suspect and aspirate the effusion and replace it with air. Only a sufficient amount of air is used to keep the pleurae from becoming adherent. The underlying lung is closely watched for the development of a parenchymal tuberculous lesion. If no pathology develops in the lung at the end of six months or a year, treatment is abandoned and complete re-expansion of the lung is allowed to take place.

In pleural effusions in which the lymphocytes are replaced by polymorphonuclear leukocytes or in which pyogenic bacteria are found, active therapy is instituted, especially if penicillin-vulnerable bacteria can be demonstrated. Our procedure has been to aspirate as much fluid as possible and instill penicillin in physiological normal sterile saline solution into the pleural space.

In those tuberculous pleurisies that occur in connection with pulmonary tuberculosis, the procedure is to aspirate the fluid, replace the aspirated fluid with air, and exert sufficient pressure on the underlying pathology to bring about control of the underlying tuberculous lesion.

The pleural effusions occurring in the course of treatment of pulmonary tuberculosis by means of pneumothorax have become less of a problem since the introduction of penicillin. The intrapleural instillation of penicillin is resorted to and its frequency of administration and dosage must depend on the laborary findings. With the sterilization of the pleural fluid and reduction in the specific gravity, the resorbing processes of the pleura become

—Continued on page 138

Vitamins in the Practice of Surgery

(Development and Application)

II*

A lack of these vital substances may occur by an insufficient supply in the food, or by impaired adsorption because of morbid alteration in the gastro-intestinal tract and bacterial destruction. Under such conditions functions connected with these factors are disturbed, when the body's own reserves have been depleted. There develop phenomena of deficiencies presenting more or less sharply defined pictures of morbidity designated respectively as avitaminoses and hypovitaminoses. As stated above vitamins and hormones cannot definitely be differentiated, considering that both groups have a common function, namely, to act as catalysts and as stimulus substances. It must not be overlooked, however, that the majority of the vitamins formed in plants cannot be synthesized by the animal organism. That is a fact which demonstrates that animal life is dependent upon the world of plants. It would mean that the vitamins are phylogenetically much older than the majority of the hormones which are formed by specific hormonal glands in the higher organized animal organisms. Thus the vitamins are looked upon as primordial substances of organic life. They are met with in the lowest living forms, such as bacteria and algae, and again we find them in animal organisms possessing neither hormonal glands nor hormones. In higher animals, where internal secretion as a physiological institution is first met with, a close relationship with the vitamins is at once established. Vitamins effecting growth in general influence development of hormonal glands and their function, entering into reciprocal reaction with hormones, now as synergists and then as antagonists. Because of these connections it is practically impossible to separate their effects, so that a lack as well as an overabundance of vitamins may appear as a hormonal effect. The relation between vitamin C and the adrenal hormones has been studied most intensively. Indeed it has been shown that vitamin C functions as a kind of stabilizer of adrenalin, that it might not be catabolized too rapidly. As regards cortin it was ascertained that the optimal effect of this hormone appears only in the presence of ascorbic acid. The phosphorylizing processes requisite for fat resorption are decisively influenced by cortin and flavin, one of the factors of the B_2 complex. A most elucidative example of an antagonism between a vitamin and a hormone is seen in the relations between vitamin A and thyroxin. Vitamin A checks the action of thyroxin and thyroxin retards excessive vitamin A effect. Results of recent investigations point more and more to the fact that the optimal activity of one group of these substances is coupled with the presence of the other. There are perhaps similar connections between vitamin B_1 and insulin, both participating decisively in the carbohydrate metabolism. It is of great importance that the effects of single vitamins, like hormones, are closely coupled with each other, and here also now in a synergistic sense and then in that of antagonism. Lack of one vitamin in a diet is of necessity followed by an alteration of the effect of other vitamins. The signs of insufficiency appearing upon the removal of one vitamin from the nutriment do not, strictly speaking, permit of a clear conception of the function of that particular vitamin within the organism. If, notwithstanding, the symptoms of deficiency are described as clinical entities characteristic of special avitaminoses, it is because under certain conditions they always present themselves in like manner, when one or the other vitamin has been removed. In the evolution of the morbid disturbances it is necessary to keep in mind that the direct loss of function of the respective vitamin is equally responsible with the altered activity of the vitamins remaining

* Part II of a series.
Being some notes made by the late H. A. H. Bouman, M.D., F.A.C.S., of Minneapolis, Minnesota.

in the food and of the hormonal functions influenced by them. When the vitamins were produced in pure form, it was shown that they could influence disease conditions which had nothing to do with avitaminoses or hypovitaminoses. Thus the vitamins possess beyond their effects as accessory food-factors also therapeutic properties. All vitamins have certain properties common to each of them, though they may vary in degree of effect according to the kind of vitamin. Generally they are almost all growth-vitamins. Furthermore all vitamins have properties, most valuable in surgical practice, which protect the body against infections. Numerous investigators have shown a definite rise of bactericidal power in blood and serum following adequate dosage of vitamin D. Of special importance for the maintenance of resistance is vitamin C. Formerly vitamin A was called the anti-infectious vitamin; it has no bactericidal effect, but insures integrity of the epithelium, the normal regeneration of which is reduced by a lack of it. Acute infections raise the vitamin requirement, so that even a normal diet may bring deficiency and thereby increase susceptibility. Excessive exposure to the sun reduces vitamin A and by radiation increases vitamin D, thus giving rise to disturbances like stone formation. Frank has shown degenerative changes in the Kupffer cells of the liver from avitaminoses, which are important agencies of defense against infection. Recently toxic diphtheria has been treated with vitamin C and adrenalin. Diphtheria toxin may be detoxicated by ascorbic acid, and Kuehnau has designated the disease as a toxic C-avitaminosis. A disease condition appearing after complete withdrawal of a vitamin is termed avitaminosis, partial removal is specified as hypovitaminosis, and the toxic syndrome produced by an overabundant supply of vitamin is called hypervitaminosis. During recent years a number of laboratory tests have been discovered which enable us to recognize a series of hypovitaminoses which are clinically non-productive of evidence.

These general remarks must suffice for orientation. It is far beyond the scope of these notes to properly discuss the source of the various vitamins, their chemistry, methods of determination, occurrence, requirements of man, physiology, pharmacology, the enormous mass of animal experimentation, commercial preparations and dosage, etc. We are specially interested in the matter of what use these vital factors might be in the practice of surgery.

(To be continued)

PULMONARY TUBERCULOSIS

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re-established. Frequently, in a very short time, a dry pleural space results.

For the purely tuberculous pleurisies where no pyogenic organisms can be found but smear or culture reveals tubercle bacilli, we have recently resorted to the use of streptomycin. The number of cases treated by this antibiotic are too few and the time since the institution of the treatment too short to draw any conclusions.

Simple thoracotomy—the open or closed drainage method for tuberculous pleurisies—often leaves permanent fistulae. Frequently, the only recourse for eradication of this complication is to resort to thoracoplasty. Such eventualities will become less frequent if the pleural effusions are treated vigorously and effectively at their onset.

In the discussion of such prosaic matters as pleural effusions, one does not become lyrical, yet it is hard not to exult at the progress of medical science. The introduction and the use of antibiotics in thoracic diseases have revolutionized the outlook and procedure. A pleural effusion whether primary or of secondary development in the treatment of pulmonary tuberculosis by means of pneumothorax is no longer the calamitous incident that it used to be. The institution of active therapy will prevent pleural thickening and unexpansion of the lung.

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SEARCH AND RESEARCH

Wallace Marshall, M. D.
Research Editor
Mobile, Alabama

VII.

Financing Your Research

This column has served the purpose, from time to time, of informing our readers as to the various phases which have to do with private research. We have attempted to call the attention of our own readers, and of others*, to the benefits which can accrue from such work. The general practitioner, along with his colleagues in the specialties, can engage in this endeavor, for it will make him a stronger clinician, along with the other benefits which he can obtain from such efforts.

We have found it essential to have access to some financial support if one expects to do this work. There are such matters as stenographic costs, postage fees, and reprint costs, which should be met when one finally publishes the results of such research. Although the outlay of money, which this involves, is not necessarily huge in amount, the researcher may find it annoying unless he possesses unlimited financial returns from his routine practice. I, for one, do not happen to be endowed with a wealthy clientele, and so it has been necessary to establish additional avenues for financial returns so that these research costs could be met.

One cannot pass a hat nor depend upon local campaigns to obtain research money. These might not be bad ideas, but our medical colleagues might object to such procedures. Hence, in order to remain in the orbit of proper decorum, it is necessary to find more ethical methods which will produce a small but welcome income which can be employed to finance research costs.

One can attempt to ask for financial grants from the various foundations. The American Medical Association assigns small grants to many researchers. But most of these workers are connected with famous clinics or universities, and the little fellow, like ourselves, might not be able to compete with such strong competition.

Not so long ago, we received a very interesting letter from a reader in California. He called our attention to the fact that many pharmaceutical houses will furnish their products to those men who seriously engage in clinical research. But we ourselves have found this practical throughout the years we have been doing clinical research. Most any reputable pharmaceutical manufacturing firm will gladly furnish their products without cost to those physicians who do conscientious clinical research. One can expect that such firms will not wish to give away hundreds of dollars worth of material if the researcher does not intend to study it adequately. Nor can one expect such a concern to furnish its products if the doctor has no intention of publishing the results of his research in a scholarly manner.

Practically all pharmaceutical firms will gladly cooperate with qualified researchers. Furthermore, these concerns have been found to be highly helpful in many other ways. Many of these corporations possess strong research staffs of their own. These men will furnish a prospective researcher with all of the available data which they possess on practically every phase of the subject which is to be investigated. They will abstract the available literature for the researcher. Many times they will even write the paper for the doctor if he will forward his research data to them. Some firms will pay for the reprint costs. Some have volunteered to mail these reprints to various doctors according to the reprint list which the physician forwards to them. We have found these pharmaceutical firms to be very important benefactors. Without them, some private research might have been impossible.

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* See our editorial in the December 1946 issue of the *American* Practitioner.

CONTEMPORARY PROGRESS

MEDICINE

Ambulatory Treatment of Peptic Ulcers with Protein Hydrolysates and Dextro-Maltose

CO TUI (*Review of Gastroenterology*, 14:108, Feb. 1947) reports 164 cases of peptic ulcer treated with protein hydrolysates and dextri-maltose. All these cases were "intractable," in that symptoms had not been relieved by any other therapeutic regimen; 144 were duodenal ulcers, 16 gastric, and 4 combined. The duration of symptoms ranged from 5 to 17 years; 102 patients showed evidence of malnutrition; 12 had hematemesis or melena; 8 a history of perforation. The protein hydrolysate is given in doses that supply 0.6 gm. nitrogen per kg. body weight; a carbohydrate, usually salt-free dextri-maltose, is added to make up 50 calories per kg. After the daily allowance of the powders is weighed out they are dissolved in a watery mixture of 1 to 1½ liters; it has been found best to make two separate solutions of the hydrolysates and of the dextri-maltose; the solutions are chilled and are usually divided into 8 equal parts to be given every 2 hours (i.e., in 16 waking hours); for patients who have night pains, an additional feeding of the protein hydrolysate, with or without the sugar, may be given when the patient is waked up by the pain. In some cases more frequent feedings are given during the day. The protein hydrolysate is given first and the dextri-maltose immediately after. For the first week no vitamins are given. The protein hydrolysate and dextri-maltose are given in place of all food until the patient is free from pain for two weeks; then one bland meal is given at breakfast time in place of one hydrolysate feeding; the next day two bland meals with 5 hydrolysate feedings. If any of these meals causes distress, however, the patient is returned to the exclusive hydrolysate feeding for another week; and the procedure is then repeated. The routine of 3 bland meals and 5 hydrolysate feedings, without dextri-maltose, is continued for two to three months. Then hydrolysates are discontinued and milk fortified with one tablespoonful of milk powder is substituted for the hydrolysate feeding, but the 2-hourly feeding schedule is continued for four to six months longer. The procedure after that becomes "indefinite" as with the other methods of medical treatment of peptic ulcer. Gastric analyses during treatment with protein hydrolysates show that the hydrolysates rapidly neutralize the gastric acidity; the hydrolysates provide an already digested product that relieves the stomach of digestive effort, and also supplies "a rich source of nutriment" that facilitates tissue repair including healing of the ulcer. The most frequent untoward effect of the treatment is diarrhea, usually painless, but this can be controlled by the administration of Kaomagma or Kaopectate. Other symptoms such as "gas pains," flushing, palpitation and headache were occasionally reported but were rare. All the patients in this series were ambulatory during treatment and usually continued to work without interruption, although a few did not work for the first week of treatment. There was relief of pain within the 24 hours after the first hydrolysate feeding in 48 cases; in 24 to 48 hours in 82 cases; and in 48 hours to one week in 18 cases. Only 16 patients did not show satisfactory immediate response to treatment. Eighteen patients were unable to continue treatment in the early part of the series, but after the protein hydrolysates and the dextri-maltose were given in separate solutions, there have been no failures due to intolerances. During the period of observation there have been 18 recurrences, but some of these patients went back on a 3 meal schedule before the end of the two months' period. The protein hydrolysate treatment, the author states, is not "a magic wand" that "banishes peptic ulcer forever" in two weeks' time. Some

follow-up regimen is necessary but what this regimen should be and how long it should be carried out is not yet determined.

Comment

The actual treatment of peptic ulcer is psychosomatic therapy plus protein in adequate quantities. Co Tui has made it simpler for the physician and patient to see that proteins are given at proper intervals. This simplification is necessary for ambulatory patients who must eat at frequent intervals. One great problem in treating these ambulatory illnesses is the question of eating away from home. Restaurants and lunch boxes are not helpful. The protein mixtures described can be prepared easily and kept in Thermos bottles.

—M.W.T.

Thiouracil in Thyrotoxicosis: Results of Prolonged Treatment in 35 Cases

EDWARD ROSE and JEANNETTE McCONNELL (*American Journal of Medical Sciences,* 213:74, Jan. 1947) report the use of thiouracil in the treatment of 35 cases of thyrotoxicosis. The dose of thiouracil used on beginning treatment was 0.6 to 1 gm. daily; the usual maintenance dose was 0.1 to 0.2 gm. daily. In 16 patients, a sustained remission of symptoms was maintained after thiouracil was discontinued for 3 to 20 months, in 9 cases for over one year. The average duration of treatment in this group was 6 months. In 4 cases in this group a second course of therapy was necessary before a sustained remission was obtained. There was only one case of nodular goiter in this group of 16 patients; in the other cases, the thyroid enlargement was diffuse and uniform. In all these cases there was complete subjective relief of symptoms, return of the basal metabolic rate and of the heart rate to normal, and gain in weight; the thyroid definitely decreased in size in 7 cases; there was no change in size of the thyroid in 8 cases, and in one case (nodular goiter), an increase in size. One patient showed a slight unilateral increase in exophthalmos. Eleven patients were maintained in remission as long as a daily maintenance dose of 0.2 gm. or less was given; in one of these cases thiouracil was stopped after 17 months therapy because of recurrent leukopenia, and subtotal thyroidectomy was done. In 2 cases remission was not maintained if the dose of thiouracil was reduced to 0.2 gm. or less; in one of these cases subtotal thyroidectomy was done because of enlargement of the goiter and increase in pressure symptoms. In 6 cases there was no satisfactory response to thiouracil. There were no definite differences in the age distribution, type of goiter, or severity of symptoms in the group in which satisfactory remission was obtained after thiouracil was stopped, as compared with the group requiring a maintenance dose of thiouracil to sustain the remission. In the group failing to respond to thiouracil, the patients were older and there was a high incidence of complicating diseases as compared with the other two groups. The toxic reactions noted were leukopenia and maculo-papular dermatosis (one case). There was only one instance in which the leukopenia was accompanied by chills, fever and pharyngitis;

thiouracil was discontinued, but was again given after one month without any toxic reaction and was continued until a sustained remission was obtained after finally discontinuing treatment. The authors are of the opinion that thiouracil may be given a trial in cases of mild and uncomplicated thyrotoxicosis if the patients are cooperative and willing to remain under close supervision; its use is also "justifiable" when patients refuse operation or are poor operative risks.

COMMENT

There are many cases where this type of therapy can be used to advantage as long as the patients are kept under close supervision. As more experience is gained from the use of thiouracil derivatives, it may be possible to make more use of this type of treatment. —M.W.T.

Folic Acid in the Treatment of Aplastic Anemia

B. R. GENDEL (*Journal of Laboratory and Clinical Medicine,* 32:139, Feb. 1947) reports 3 cases of aplastic anemia treated with folic acid. The value of folic acid in the treatment of pernicious anemia and other macrocytic anemias has recently been demonstrated in a number of cases, but the results reported with folic acid in aplastic anemia have been disappointing. In experiments on rats, however, it has been shown that deficiency of folic acid causes an aplastic bone marrow. In the 3 cases reported the blood picture showed the pancytopenia characteristic of aplastic anemia; in 2 of the patients the bone marrow, as shown by sternal marrow aspiration, was hypoplastic, although in one case it was slightly hyperplastic. In the first case in which folic acid was used, weekly blood transfusions were necessary to maintain the red cell count at 2.5 millions, and the patient was having frequent transfusion reactions. In this case folic acid in doses of 200 mg. daily (increased to 400 mg. daily for two weeks) resulted in a satisfactory remission, better than that primarily obtained with transfusions; and a marked improvement in the patient's general condition. In the second case, the use of folic acid, also in large doses, made it possible to dispense with repeated blood transfusions, and maintain the blood count at the same level, although the blood picture showed no definite improvement. In the third case, the patient had a partial remission before beginning treatment with folic acid; but the red cell count remained low and showed no improvement under treatment with iron and liver extract. A good remission was obtained with folic acid therapy (200 mg. daily) and was maintained for eight months after treatment was discontinued. On the basis of these results the author recommends that in cases of aplastic anemia, treatment with folic acid in large doses used over a long period of time should be given a trial.

COMMENT

Certainly a welcome treatment for aplastic anemia and should be tried extensively. —M.W.T.

Gastragogue Effect of Laxatives and Allyl-Bromide Mixture

Defining a gastragogue as a substance that aids in emptying the stomach of its contents, H. M. FEINBLATT and E. A. FERGUSON (*American Journal of Digestive Diseases,* 13:386, Dec. 1946) reported their clinical and roentgenographic study of the allyl-bromide mixture consisting of all the natural products of bromination of garlic. The allyl-bromide mixture was demonstrated to be a useful agent in stimulating gastric motility and physiological emptying of the stomach which shows retention. The gastragogue effect was associated with subjective relief of symptoms. When allium sativum is brominated, the resulting allyl-bromide mixture shows the clinical action of a carminative. Gastric analyses on rats showed definite evidence of gastric stimulation. The laxative drugs studied did not increase stomach motility or secretion.

COMMENT

It is interesting to note that laxatives do not increase stomach motility. There are many aged persons who could be given a trial of the allyl-bromide mixture. Many of these patients have gastric retention without any apparent cause. They are distressed especially at night.—M.W.T.

Renal Complications of Sulfonamide Therapy

B. S. ABESHOUSE and L. M. TANKIN (*Journal of Urology,* 56:658, Dec. 1946) report 12 cases of renal complications following sulfapyridine therapy, 12 cases following sulfathiazole therapy, 14 cases following sulfadiazine therapy and 2 cases following sulfamerazine therapy. Renal complications following sulfonamide therapy are of two types: mechanical, caused by the deposition of crystals or concretions in renal tubules, pelvis, or ureter; and chemical, acute toxic and degenerative nephritis. Every person receiving any sulfonamide should be under close observation for the detection of any symptom of renal complication. Crystalluria alone does not indicate serious renal damage, but is an indication for temporary withdrawal of the drug, or decreasing dosage, forcing fluids and alkalinization. Such symptoms as hematuria, anuria or oliguria, azotemia and renal or abdominal pain indicate immediate withdrawal of the drug, and prompt institution of treatment. In anuria, cystoscopy or ureteral catheterization may be necessary to differentiate between complications of the mechanical type and of the chemical type. If the usual conservative measures and catheterization fail to restore function, operation may be necessary, either decapsulation alone or combined with pyelostomy or nephrostomy or restrograde ureteral catheterization. In the authors' 12 cases of renal complications following sulfapyridine therapy, hematuria was a symptom in every case; renal colic was present in 2 cases; urolithiasis, acetonuria, anuria in one case each. Conservative therapy was successful in every case, and cystoscopy was not necessary. In the 12 cases of complications following sulfathiazole therapy, hematuria was a symptom in 7 cases, uncomplicated in 3 cases, associated with azotemia, crystalluria, oliguria, anuria in one case each; crystalluria was the only symptom in 2 cases and anuria in 3 cases. Cystoscopy and ureteral catheterization were necessary in 3 cases; in the others conservative therapy resulted in recovery. In the 14 cases of complications following sulfadiazine therapy, hematuria was a symptom in 12 cases, without other symptoms in 2 cases, with renal colic and azotemia in 4 cases, with crystalluria, renal stone, oliguria, anuria in one case each; oliguria and azotemia were present in 2 cases; crystalluria in 2 cases, and anuria in one case. There was one death in this group in which anuria failed to respond to decapsulation and nephrostomy; in this case autopsy showed thrombosis of both renal arteries, a complication not previously reported following sulfonamide therapy. In the 2 cases of complications following sulfamerazine therapy, anuria was present in both, with hematuria in one case and renal pain in the other. One responded satisfactorily to conservative therapy, the other required ureteral catheterization. Renal complications of sulfanomide therapy can be prevented or their incidence reduced by the following measures: determining previous sulfonamide therapy, drug sensitivity or idiosyncrasy; determination of renal function before and during therapy; use of accurate dosage; maintaining water balance; alkalinization to sustain the pH at 76 in the urine.

COMMENT

Two sentences cover the ground. The sulfonamides are all very dangerous and no precaution is unnecessary.—V.C.P.

Treatment of Azoospermia by Vaso-Epididymal Anastomosis

LEWIS MICHELSON (*Western Journal of Surgery, Obstetrics and Gynecology,* 55:120, Feb. 1947) in a study of 1083 cases of sterile marriages found azoospermia (absence of spermatozoa in the semen) in the male in 146 cases, 13.5 per cent. Azoospermia may be due to defective production of spermatozoa or it may be due to obstruction of the passageways for them. Operation for reconstruction of the obstructed passageways is indicated only when a normal testicle is present and when the blockage of the ductal system is not so extensive as to make restoration of potency impossible. The presence of a normal testicle is determined by physical

examination and testicular biopsy. Fibrosis of the duct sufficient to cause blockage may be recognized by physical examination, but as a rule the extent of the disease process in the duct system can be determined only at operation. In the author's series of 146 cases the azoospermia was found to be due to blockage of the ducts in 60 cases. The most common cause of this blockage was gonorrheal vaso-epididymitis, in 35 cases. In 23 of these cases the disease caused blockage of both sides, and in 12 cases blockage of one side. In one case absence of spermatozoa from the other testicle was due to cryptorchidism and in 2 cases blockage of the opposite side was due to trauma; in the other 9 cases, the cause of the nonpotency of the other side could not be determined. Trauma was the cause of the obstructive lesion in 25 cases, involving both sides in 20 cases, and one side in 5 cases. In 2 of these 5 cases, blockage of the opposite side was due to gonorrheal vaso-epididymitis and in the other 3 cases the cause could not be determined. There were 4 cases of congenital absence of both vasa; testicular biopsy showed normal spermatogenesis in these cases. Operation for vaso-epididymal anastomosis was done in 8 patients, in 6 of whom blockage of the ductal system was due to gonococcal infection and in one to trauma; in one case the etiology was unknown. The anastomosis was done on both sides in 3, and on one side in 5 patients. Spermatozoa were found in the semen after operation in 3 cases (2 unilateral, one bilateral anastomosis); the wives of 2 of these 3 men have become pregnant (one bilateral, one unilateral anastomosis). The operation employed is described in detail. It is a modification of the Hagner-Martin operation but introduces a new principle by producing a permanent fistula in the anastomotic opening between the vas and the epididymis by the use of stainless steel wires running through the lumen of the vas and the epididymal tubule, which are removed in ten to fourteen days.

COMMENT

This operation is very inviting. The one point not covered is: for how long do the promising results persist? It is very easy for postoperative changes in the annexa to cancel the operation out.—V.C.P.

Transurethral Resection and the Paraplegic

H. C. BUMPUS, JR. (*Journal of Urology*, 57:300, Feb. 1947) reports his experience in a service especially organized for patients with cord bladder due to spinal cord injury. After several months, usually in four to five months, the majority of paraplegic patients develop automatic bladders; during the period while automaticity is developing the use of a suprapubic drain is superior to a permanent urethral catheter, as it avoids the dangers of infection and pressure. In a group of 87 patients with cord bladder, 58 developed automatic bladders; in 37 of these the action of the bladder is fully efficient, but in 16 of these cases, removal of tissue from the bladder neck was necessary before the bladder could function adequately. If with an automatic bladder only one or two ounces of residual urine are present and the urine is clear, transurethral resection is not considered to be necessary. The operation is done only when many ounces of residual urine remain after the bladder has contracted automatically or when the patient is unable to expel any urine. In most cases of traumatic lesions of the spinal cord there is more or less spasticity of the bladder muscles and ureteral sphincters; the satisfactory emptying of the bladder depends on the ability of the detrusor muscles to overcome any obstruction exerted by the sphincter muscles. If this obstruction is not satisfactorily overcome, transurethral resection removes the obstruction and enables both the automatic and the voluntary types of paraplegic bladder to function. As in paraplegic patients healing is slow and there may be late bleeding after transurethral resection, owing to the spasticity of the muscles, only a minimum amount of tissue is removed in these cases, even if a second or third resection is necessary. Three resections were done in 3 of the author's cases, but even in these cases the total amount of tissue removed was small, rarely over 10 gm. Only 6 of the author's patients on the

Cord Bladder Service at the Corona Naval Hospital were using a catheter to empty the bladder at the time of this report; 33 patients not only empty their bladders, but also pass sterile urine.

COMMENT

This study deserves careful attention on account of the recovery, at least in part, of many of these difficult and unpromising cases.
—V.C.P.

Experience in the Extraction of Ureteral Calculi with a Balkus Looped Catheter

J. J. NUGENT (*Southern Medical Journal*, 40:26, Jan. 1947) reports the use of the Balkus looped catheter for extraction of ureteral stones for a period of two years. In this period, the looped catheter was the first instrument introduced whenever a ureteral calculus was suspected. It was found to be important to avoid twisting or rotating the catheter during attempts to pass any obstruction. In the two-year period, ureteral calculi have been successfully removed with this catheter in 23 cases requiring 29 cystoscopies. In one case, a stone shown by the x-ray to be low in the renal pelvis was extracted with this catheter. In 8 cases, the calculus was not extracted, although the catheter was passed beyond it. In 5 cases no ureteral instrument could be passed beyond the calculus. Five urelithotomies were done in this two-year period; and approximately 40 patients passed ureteral calculi spontaneously. The introduction of the catheter and forming of the loop has caused little pain; the patient usually complains of pain during the withdrawal of the loop, even if a stone is not present. All patients have been given seconal as a sedative, and pentothal sodium anesthesia has been frequently used.

COMMENT

In the old days and not in urgent cases we passed a progressively larger and larger bougie up to the stone. Gentle pressure was used to force the stone back and beyond the stricture commonly present. After a while the stone usually passed spontaneously.
—V.C.P.

✚

SEARCH AND RESEARCH

—Concluded from page 139

The beginner in research has to prove that he is honest in such work. His data must be presented in a truthful manner. These pharmaceutical and chemical firms are honest and most reliable. When they have received proof that the private investigator is conscientious, they will usually support his research in a munificent manner. I could not have accomplished much research without such assistance, and therefore I desire to bring this to the attention of my readers who wish to do clinical research.

One does not have to feel that his research project will be lifted if he is asked to present his plan of procedure to an ethical firm. Such data are guarded zealously, and are treated in a highly confidential manner.

If you have sound ideas which you wish to develop, and the costs of research seem to be insurmountable, it might be advisable to ask the assistance of any reputable pharmaceutical firm which manufactures products for use in the particular field under study. Most of these firms employ a medical director who is very well acquainted with most problems which have to do with such research. I have found these men to be brilliant, ethical individuals who are happy to assist most physicians with their research problems.

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Medical BOOK NEWS

Edited by
ALFRED E. SHIPLEY, M.D., Dr. P.H.

All books for review and communications concerning Book News should be addressed to the Editor of this department, 1313 Bedford Avenue, Brooklyn 16, N. Y.

JEAN-BAPTISTE BOUILLAUD
1796 ~ 1881

Classical Quotations

● The narrowing of the orifices of the heart, following different kinds of thickening and induration of the valves, is the gravest anatomic accident that endocarditis may carry in its wake . . . The degrees of narrowing of the orifices of the heart are very variable. In extreme degrees, one can hardly introduce the tip of the little finger, or even the tip of a writing pen, into the narrowed orifice.

JEAN BAPTISTE BOUILLAUD

Traite Clinique des Maladies du Coeur, Paris, 1835. Section Deuxieme, Histoire Generale de l'Endocardite, Article Premier.

Psychological Medicine

Psychological Medicine. A Short Introduction To Psychiatry. With an appendix on PSYCHIATRY ASSOCIATED WITH WAR CONDITIONS. By Desmond Curran, M.B., Eng., & Eric Guttmann, M.D. 2nd Edition. Baltimore, Williams & Wilkins Co., [c. 1945]. 8vo. 246 pages, illustrated. Cloth, $3.50.

THIS second edition is some 58 pages longer than the first with considerable revision and additions, especially in connection with sections on constitutional factors, psychopathic personalities, affective and hysterical syndromes.

The legal aspects, although not appropriate to American practice, are worthy of perusal. The appendix on war psychiatry, and the examination, management and treatment of service cases is practical and pithily formulated.

The general practitioner and medical student will find herein a salutary example of common-sense psychiatry at its best.

FREDERICK L. PATRY

Medico-Sociological Problems

A Surgeon Looks at Life. By Richard A. Leonardo, M.D. New York, Froben Press, [c. 1945]. 8vo. 128 pages. Cloth, $2.00.

THIS book can be recommended to the public. The point of view is conservative, with prevailing medical opinions given. No radical personal views are presented. The author has done wide reading and presents strikingly interesting facts. Most of the book is devoted to modern up-to-the-minute medical and sociological problems.

C. W. HENNINGTON

Ancient Medical Science

Hippocratic Wisdom. For Him Who Wishes to Pursue Properly the Science of Medicine. By William F. Petersen, M.D. Springfield, Ill., Charles C. Thomas, [c. 1946]. 8vo. 263 pages, illustrated. Cloth, $5.00.

THE physician or surgeon, who prides himself on his modern knowledge will be impressed with this book. Modern knowledge will turn out to be the same as held by Hippocrates. The symptomatology of many well known diseases of our times was clearly recorded long ago.

The author of this book had keen delight in preparing the text, and all those who enjoy reading the foundations of medical science are promised several hours of interest and pleasure.

J. ARTHUR BUCHANAN

Who Discovered the Circulation?

The Circulation of the Blood and Andrea Cesalpino of Aresso. By Dr. John P. Arcieri. New York, S. F. Vanni, [c. 1945]. 8vo. 193 pages, illustrated. Cloth, $4.00.

IN quaint English the professor of medical history in the University of Rome brands William Harvey as an unscrupulous plagiarist. Cesalpino's book antedated *De Motu Cordis* by more than fifty years. Many British authorities are roughly handled. A good case is made out.

CHARLES A. GORDON

Hunger

The World's Hunger. By Frank A. Pearson & Floyd A. Harper, Ithaca, N. Y., Cornell University Pr., [c. 1945]. 8vo. 90 pages. Cloth, $1.50.

THIS treatise states in simple, easily understood terms the problem of feeding the world and the natural limitations involved in an attempt by a country such as the U.S.A. to raise the standards of living of less fortunate countries to "par." "There is (in the world) no scarcity of land with favorable topography or adequate sunlight, and carbon dioxide or favorable temperature or reliable rainfall, or fertile soil, or adequate rainfall. There is, however, a serious shortage of land with the *proper combinations* of these seven factors that are so necessary for food production." The authors prove their theory adequate by data relative to the seven factors over the surface of the world. We Americans may well be cautious and remember that Charity should begin at home.

GEORGE E. ANDERSON

Food

The Microbiology of Foods. By Fred Wilbur Tanner, D.Sc. 2nd Edition. Champaign, Ill., The Garrand Press, [c. 1944]. 8vo. 1196 pages. Cloth, $12.50.

THE second edition of this book is complete, and up to date. The author has described methods, culture media, and procedures of tests for analyzing and interpreting findings in examining various kinds of foods. Each chapter is rich with reference material. This book is a necessity for any physician, public health or industrial laboratory that has to do with foods or food handling.

CASPAR G. BURN

Psychotherapy

Group Psychotherapy. Theory and Practice. By J.W. Klapman, M.D. New York, Grune & Stratton, [c. 1946]. 8vo. 344 pages, illustrated. Cloth, $4.00.

THIS comprehensive and instructive review of psychotherapy is divided into three parts: Historical and anthropological considerations of group psychology and psychotherapy, the dynamics and theory of group therapy, and methods of administration.

It is a practical exposition of the administration of group therapy as well as a scholarly adaptation of psycho-analytical technics to group diagnosis and re-education. This volume should be a valuable textbook for all psychiatrists and psychologists and a good source moterial for students and religious teachers.

C. MILTON MEEKS

Psychosomatic Therapy

The Person in the Body. 3y Leland E. Hinsie, M.D. New York, W. W. Norton & Co., [c. 1945]. 8vo. 263 pages. Cloth, $2.75.

THE author tries to present within its pages an introduction to psychosomatic medicine. We agree with him when he says "The passing of the family physician is to be regretted, since it was he who knew the emotions of his patients; . . . he recognized what roles the physique played in disrupting their daily life; he knew the strength and weakness of their feelings. He prescribed for both."

The author's emphasis on this point is worth while. The preservation of the personal physician—patient relationship provides an uninterrupted psychosomatic therapy.

The work seems to stress the Freudian conception of sex interpretation in psychosomatic evaluations.

MORRIS ANT

Veterans' Adjustments

Back to Life. The Emotional Adjustment of Our Veterans. By Herbert I. Kupper, M.D. New York, L. B. Fischer Publishing Corp., [c. 1945]. 12mo. 220 pages, Cloth, $2.50.

DR. KUPPER, staff psychiatrist of the United States Marine Hospital, Ellis Island, New York, has performed a meritorious service in writing this straightforward, lucid account of the travels and travails of the soldier from induction station to his return to civilian status. It does not read like a textbook although its contents are so thoroughly organized that it could very well serve as a text for all those who are more or less responsible

for the understanding, treatment and rehabilitation of the returning serviceman and woman. One is never left with the feeling that the author is discussing men and women in the abstract, or mere "cases" which are found in goodly number, but rather individual human accounts of the actual problems which are found in the mind of the servicemen as well as those facing physicians, psychiatrists, psychologists, social workers, educational and vocational counsellors, educators, employers, members of their families and all those who are in their circle of friends and acquaintances.

Of the several books published to date which have had as their purpose the objectives of this book, there is none to the reviewer's mind at least, which presents the problems so readably and lucidly, and at the same time clearly formulates methodology to subserve these ends.

FREDERICK L. PATRY

Diet and Health

Nutrition and Physical Degeneration. A Comparison of Primitive and Modern Diets and Their Effects. By Weston A. Price, D.D.S. Redlands, Calif., The Author, [c. 1945]. 8vo. 527 pages, illustrated. Cloth, $5.50.

THIS book shows the effects of painstaking study and research. The discussion on all primitive diets and their relationship to health paralleled with the discussion on civilized dietary and its relation to health leads to the author's opinion that refined foods are the cause of tissue degeneration and poor health.

The supplement discusses an X factor in butter fat; fluorine in plant and animal life and the effect of good soil on the mineral and vitamin content of food. The author stresses that these factors if properly employed will aid in the prevention of dental caries, and in maintaining good tissue resistance.

In the light of present interest in nutrition, this book is highly recommended.

MORRIS ANT

BOOKS RECEIVED

for review are promptly acknowledged in this column: we assume no other obligation in return for the courtesy of those sending us the same. In most cases, review notices will be promptly published shortly after acknowledgment of receipt has been made in this column.

Charts for the Calculation of Environmetal Warmth. Supplement to War Memorandum #17. London, His Majesty's Stationery Office, [1946]. Folio. Series of 10 charts.

Spuren von Stoffen Entscheiden Uber Unser Shicksal. By Professor Emil Abderhalden. 2nd Edition. Basel, Switzerland, Benno Schwabe & Co., [c. 1946]. Large 12mo. 106 pages, illustrated. Paper, 6 fr.

A Primer for Diabetic Patients. By Russell M. Wilder, M.D. 8th Edition. Philadelphia, W. B. Saunders Co., [c. 1946]. 16mo. 192 pages, illustrated. Cloth, $1.75.

Jewish Luminaries in Medical History. By Harry Friedenwald, M.D. Also a CATALOGUE OF WORKS BEARING ON THE SUBJECT OF THE JEWS AND MEDICINE FROM THE PRIVATE LIBRARY OF HARRY FRIEDENWALD. Baltimore, Johns Hopkins Pr., [c. 1946]. 8vo. 197 pages, illustrated. Cloth, $3.00.

X-Rays and Radium in the Treatment of Diseases of the Skin. By George M. MacKee, M.D. & Anthony C. Cipollaro, M.D. Contributor, Hamilton Montgomery, M.D. 4th Edition. Philadelphia, Lea & Febiger, [c. 1946]. 8vo. 668 pages, illustrated. Cloth, $10.00.

Description and Measurement of Personality. By Raymond B. Cattell. Yonkers, N. Y., World Book Co., [c. 1946]. 8vo. 602 pages. Cloth, $4.00. (Measurement and Adjustment Series).

Dentistry. An Agency of Health Service. By Malcolm Wallace Carr, D.D.S. New York, Commonwealth Fund, [c. 1946]. 8vo. 219 pages. Cloth, $1.50.

Mongolism and Cretinism. A Study of the Clinical Manifestations and the General Pathology of Pituitary and Thyroid Deficiency. By Clemens E. Benda, M.D. New York, Grune & Stratton, [c. 1946]. 8vo. 310 pages, illustrated. Cloth, $6.50.

Principles of Hematology. By Russell L. Haden, M.D. 3rd Edition. Philadelphia, Lea & Febiger, [c. 1946]. 8vo. 366 pages, illustrated. Cloth, $5.00.

Practical Malariology. Prepared Under the Auspices of the Division of Medical Sciences of the National Research Council. By Paul F. Russell, M.D., Luther S. West, Ph.D. & Reginald D. Manwell, Sc.D. Philadelphia, W. B. Saunders Co., [c. 1946]. 8vo. 684 pages, illustrated. Cloth, $8.00.

R K G Rheocardiography. A Method of Circulation's Investigation and Diagnosis in Circular Motion. By Dr. Wolfgang Holzer, Dr. Kurt Polzer & A. Marko. Translated by Mrs. Emma M. Kreidl. Vienna, Wilhelm Maudrich, [c. 1946]. 8vo. 43 pages, illustrated. Paper, sfr. 7.

Medical Uses of Soap. A Symposium. Edited by Morris Fishbein, M.D. Philadelphia, J. B. Lippincott Co., [c. 1946]. 8vo. 195 pages, illustrated. Cloth, $3.00.